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DISSERTATION

ON

SPONTANEOUS BACTERIAL PERITONITIS IN PATIENTS WITH CIRRHOSIS LIVER WITH ASCITES

SUBMITTED FOR M.D. DEGREE EXAMINATION

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CERTIFICATE

This is to certify that this dissertation entitled **"SPONTANEOUS BACTERIAL PERITONITIS IN CIRRHOSIS LIVER WITH ASCITES"** submitted by **Dr. A. C. SASI KUMAR** to the faculty of Internal Medicine, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.D., degree Branch I (Internal Medicine) is a bonafide research work carried out by him , under my direct supervision and guidance.

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CONTENTS

INTRODUCTION

Spontaneous Bacterial Peritonitis is an infectious process that usually occurs in the cirrhotic patients with ascites, in which a contiguous local source of infection is absent. The diagnosis of Spontaneous Bacterial Peritonitis is made, when there is a positive ascitic fluid culture (always monomicrobial) and there is an elevated ascitic fluid absolute neutrophil count (at least 250 cells / mm^3 or above).

Spontaneous Bacterial Peritonitis is a common complication occurring in cirrhotic patients, mostly fatal in nature, if left untreated. The mortality ranges from 48% to 57%[1.2]. Spontaneous Bacterial Peritonitis is defined as an infection of the ascitic fluid in the absence of any intra-abdominal surgically treatable source of infection. The prevalence varies from 8% to 27% [3, 4].

The higher awareness of this entity over the past two decades has decreased the threshold for performing diagnostic procedures and has resulted in the large increase in the number of patients so diagnosed.

Though a large amount of information on Spontaneous Bacterial Peritonitis is available today, the condition is however under diagnosed because of the following facts,

(i) The protein concentration of ascitic fluid does not increase during Spontaneous Bacterial Peritonitis in contrast to other infected body fluid[5].

(ii) 35% to 58% of patients with Spontaneous Bacterial Peritonitis are culture negative by conventional methods.[6, 7]

Recurrence of Spontaneous Bacterial Peritonitis occurs in 70% within one year after recovery from the first episode.[2]

AIM OF THE STUDY

1. To study the prevalence of Spontaneous Bacterial Peritonitis in cirrhotic patients with ascites.

2. To study the presenting features of Spontaneous Bacterial Peritonitis.

3. To study the correlation of severity of liver disease with Spontaneous Bacterial Peritonitis.

4. To study the correlation of ascitic fluid chemistry and cytology with Spontaneous Bacterial Peritonitis.

5. To compare the culture of ascitic fluid using blood culture bottles with that of conventional method of culture.

6. To study the microbiological profile of Spontaneous Bacterial Peritonitis.

REVIEW OF LITERATURE

Though described first in 1893 by French physicians **CHARRIN and VEILLON,** wide spread recognition came about only in mid 1960's and 1970's. Numerous retrospective [2,3,8,9] and two prospective studies[1] have confirmed the importance and prevalence of the Spontaneous Bacterial Peritonitis in cirrhotic patients with ascites.

In the seven papers, which have reported more than 20 cases each of Spontaneous Bacterial Peritonitis[2, 3, 8, 9] 66% of 246 patients were reported to be alcoholics and of 177 patients who had histologic evaluation, 68% had alcoholic cirrhosis. However, cirrhosis is not the only condition, which predisposes to Spontaneous Bacterial Peritonitis. This condition was the most common cause of death in a childhood series of nephrotic syndrome reported in 1950. There are also case reports of Spontaneous Bacterial Peritonitis in patients with severe viral hepatitis, CCF and malignancy related ascites.

Ascitic fluid infections can be spontaneous or secondary to an intra-abdominal, surgically treatable source of infection. More than 90% of ascitic fluid infections are spontaneous.

According to the characteristics of ascitic fluid culture and neutrophil count, three other variants of ascitic fluid infection have been described in cirrhotic patients during the last decade.

(i) Culture negative neutrocytic bacterascites (culture negative neutrocytic ascites).

(ii) Mono-microbial non-neutrocytic bacterascites.

(iii) Poly-microbial bacterascites.

VARIANTS OF ASCITIC FLUID INFECTIONS ACCORDING TO ASCITIC FLUID CHARACTERISTICS

Sl.No	VARIANTS OF A.F.I	A.F.CULTURE	A.F. PMN count/mm^3
1	Spontaneous Bacterial Peritonitis	+	≥ 250
2	Culture negative neutrocytic ascites	_	≥ 250
3	Mono-microbial non-neutrocytic ascites.	+	< 250
4	Secondary bacterial peritonitis	+ (Single/ multiple org.)	≥ 250
5	Poly-microbial bacterascites.	+ (Multiple org.)	< 250

Culture negative neutrocytic ascites is defined as ascitic fluid infection in which the neutrophil count is 250 or more with no growth of ascitic fluid culture in the absence of prior antibiotic therapy and in the absence of another explanation for an elevated neutrophil count, such as peritoneal carcinomatosis, tuberculosis or pancreatitis[6]. 34% of culture negative neutrocytic ascites converted to a culture positive infection before antibiotic therapy was started, where as 66% of patients remained culture negative, usually with a spontaneously declining neutrophil count.

These data suggest that some episodes of culture negative neutrocytic ascites can resolve spontaneously. Therefore, culture negative neutrocytic ascites must be considered as a true infection of ascitic fluid and it must be treated with appropriate antibiotics.

Mono-microbial non-neutrocytic ascites is defined as an ascitic fluid infection in which neutrophil count is less than 250 cells/mm^3 with a positive ascitic fluid culture for a single organism.[10] It was observed that Mono-microbial non-neutrocytic ascites episodes resolved spontaneously in 62% to 86% of patients. [10, 11] All patients who progressed to spontaneous bacterial peritonitis had symptoms of bacterial infection at the time of first paracentesis. Therefore, only symptomatic patients with Mono-microbial non-neutrocytic ascites appear to require treatment with antibiotics. Whereas asymptomatic patients should be treated or reevaluated with a second tap before antibiotic treatment is started. Patients with bacterascites have less severe liver disease than patients with spontaneous bacterial peritonitis. [10, 11, 12]

In summary, spontaneous ascitic fluid infection is a very dynamic process. Bacteria can resolve spontaneously or progress to spontaneous bacterial peritonitis. Although occasionally spontaneous bacterial peritonitis can spontaneously evolve to sterile fluid with a declining neutrophil count, this effect is unusual and it cannot be relied on or predicted. An elevated ascitic fluid neutrophil count probably provides evidence that peritoneal macrophages have failed to contain bacterial colonization and that neutrophils have to be called in as the second line of defence.[13] Without antibiotic treatment, neutrophils usually fail with resulting uncontrolled infection and

death of the host. An ascitic fluid neutrophil count of 250 or more should be presumed to be caused by bacterial infection and it should be treated empirically with antibiotics.

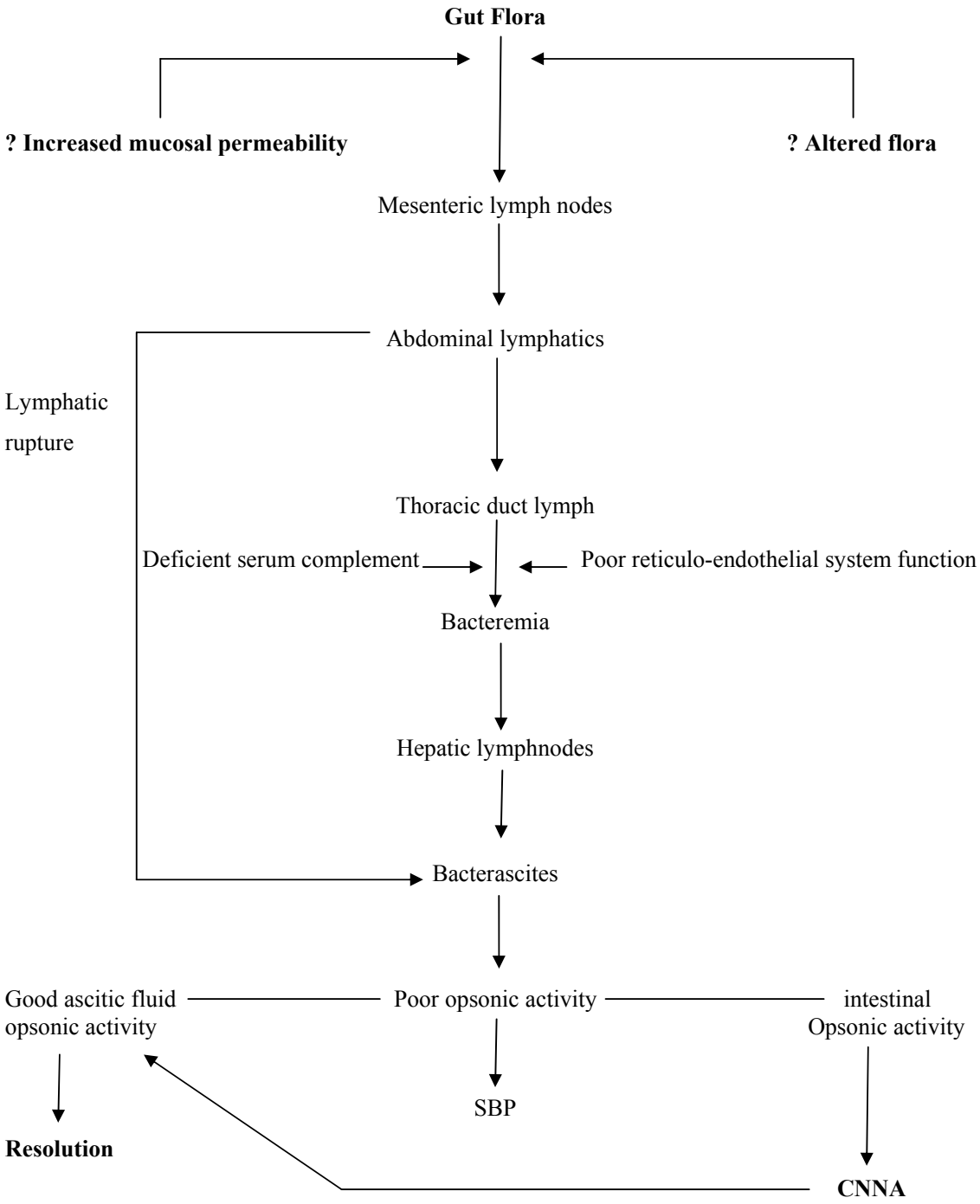
Secondary bacterial peritonitis can also develop in the cirrhotic patients. Differentiation of spontaneous bacterial peritonitis from secondary bacterial peritonitis is very important, because SBP is always treated medically and secondary bacterial peritonitis is treated surgically. Secondary bacterial peritonitis is usually suspected when neutrocytic ascitic fluid demonstrates two or three of three of the following criteria.[14] Total protein level greater than 1 gm/dl, Glucose level less than 50mg/dl, and LDH level greater that 225mu/ml.

Further more, most ascitic fluid cultures in these patients are poly-microbial, whereas in Spontaneous Bacterial Peritonitis they are mono-microbial.

Poly-microbial bacterascites is another variant of non specific ascitic fluid infection. This is characterized by (i) a neutrophil count of less than 250 cells/mm^3 and multiple organisms on gram stain and culture. (ii) as a result of needle perforation of the gut during attempted paracentesis. Poly-microbial bacterascites can resolve spontaneously without antibiotic treatment, if ascitic fluid protein (and opsonin) concentration is adequate.

FLOW CHART – I

PATHOGENESIS OF SBP



PATHOGENESIS OF SPONTANEOUS BACTERIAL PERITONITIS

PHYSIOLOGY OF PERITONEUM

Peritoneum is a complex serous membrane, which lines the abdominal wall and is reflected over the viscera within the abdomen. The parietal and visceral layers are developed respectively from the somatopleural and splanchnopleural layers of the lateral plate mesoderm.

The total area of the peritoneal surface in the adults is between 1.5 and $2m^2$ approximately equal to the total body surface area. The blood flow to the peritoneum is 50 to 70ml/min. The normal peritoneum consists of a single layer of flattened mesothelial cells. Micro-villi protrude from the free mesothelial surface which is lubricated by a small volume of serous fluid.

PATHOGENESIS

Pathogenesis of SBP in patients with cirrhosis is considered to be the main consequence of Bacterial Translocation (BT).

There are some mechanisms that are being proposed to explain BT in cirrhosis,

(1) The intestinal bacterial overgrowth,

(2) The structural and functional alterations of the intestinal mucosal barrier and

(3) The deficiencies of the local immune response[15, 16].

The intestinal bacterial overgrowth plays a key role in BT in cirrhosis and is the result of the delayed intestinal transit existing in these patients. It seems that the sympathoadrenal stimulation, increases NO synthesis and the oxidative stress of the mucosa are the main causes for decreased intestinal motility[17,18].

The barrier of the intestinal mucosa includes defence mechanisms of secretory or physical type, against the microbial penetration.

The secretory (first defence) mechanism is realized through the mucus secretion, the local immunoglobulins and the bile salts.

The physical (second defence) mechanism is represented by the intestinal epithelium-by its lack of permeability and its antimicrobial peptide active production.

Among the predisposing factors for SBP, the most important one is the severity of liver diseases: about 70% of the patients who develop SBP are in Child C class. Besides, a serum bilirubin level > 2.5 mg/dl is an independent predictive factor of SBP[19].

20% of the patients have SBP at the time of admission to the hospital and 30-40% develop bacterial infections, during hospitalization for GI hemorrhage – a possible explanation being that the hemorrhagic shock increases BT and intestinal permeability. Also for preventing bacteremia, vascular catheterization has to be reduced to the minimum.

For any infection, there are source of organisms and conditions predisposing to the infection. The presence of ascites alone is not sufficient to result in spontaneous

bacterial peritonitis, since not all forms of ascites are prone for infection. Cirrhotic ascites is similar to most other types of ascites except in its low protein concentration. Cirrhosis is also associated with poor clearance of substances from the blood including bacteria.

Spontaneous Bacterial Peritonitis develops in patients with advanced liver disease[3]. It is a consequence of multiple defects in the immune defence of patients with cirrhosis. Patients with liver disease have deficient serum and ascitic fluid complement levels, [20, 21, 22] decreased reticulo-endothelial system phagocytic activity, [23, 24] alterations in neutrophil functions [25] and diminished bactericidal and opsonic activity of ascitic fluid.[26, 27, 28, 29]

SOURCE OF BACTERIAL INFECTION AND MODE OF ENTRANCE TO THE PERITONEAL CAVITY

Most cases of spontaneous bacterial peritonitis are not associated with an established infection elsewhere in the body and therefore infection in the peritoneal fluid must arise from the blood stream or across the intestinal wall. 75% of the organisms causing spontaneous bacterial peritonitis are intestinal flora and both mechanisms have been implicated. Evidence favouring a trans-intestinal wall route includes the presence of mucosal edema and the demonstration of transfer from lumen to the peritoneal cavity of both alive and dead C_{14} labelled E.coli. However the association of spontaneous bacterial peritonitis with poor clearance of particulate matter by reticulo-endothelial cells and with spontaneous bacteremia supports a

bloodborne route, as does infection with organism that does not inhabitat the gut. Most non-enteric organisms probably enter the ascitic fluid by one of the two following mechanisms,

(i) Across the sinusoids of liver with subsequent transfer into ascitic fluid across the hepatic capsule or

(ii) Across the capillaries of the intestine with subsequent direct anatomic breaks in lymphatics, drawing the infected intestinal fluid.

The normal intestinal capillary permeability and "Capillarisation of the Sinusoids" that occurs with cirrhosis would suggest that the quantity of organisms entering either route is small[30].

INTRAHEPATIC SHUNTING AND SYSTEMIC CLEARANCE OF BACTERIA

Spontaneous bacteremia is frequent in cirrhosis. The type of organisms in Spontaneous bacteremia is primarily enteric and similar to those found in spontaneous bacterial peritonitis. Both syndromes are compatible with the poor first pass removal of gut bacteria. The systemic clearance of substances removed by reticulo-endothelial cells and by hepatocytes, decreases in parallel with the severity of chronic liver disease and is thought to be due to the hepatic fibrosis producing capillarisation of the sinusoids with acinae, septal intrahepatic shunts or both. Capillarisation of sinusoids is the source of deposition of collagen in the space of Disse and sinusoids within the

acinae. This results in an abnormal distribution of acinar blood flow and the separation of hepatocytes from the blood stream. Septal intra-hepatic shunts are not connected with normal acinae since they completely bypass the acinar unit. Capillarisation of sinusoids is probably the most frequent causes of intra-hepatic shunting in patients with chronic liver disease. Septal intra-hepatic shunts are normally greater than 25 microns in diameter and can be measured by the portal injection of microspheres. Such intra-hepatic shunts increase the exposure of the systemic circulation to the gut products. Since the first pass hepatic clearance is decreased and the clearance of these products in the systemic circulation is also decreased, intra-hepatic shunting may be a major factor predisposing to spontaneous bacterial peritonitis[23].

CHARACTERISTICS OF ASCITES PREDISPOSING TO INFECTION

Spontaneous bacterial peritonitis in cirrhotic patients is rare, in the absence of ascites. Even in the patients in whom clinically detectable ascites appear to follow after the evidence of spontaneous bacterial peritonitis has developed, a small amount of ascites must have been present prior to the infection. The rarity of the spontaneous bacterial peritonitis in all types of ascites, except secondary to liver disease, may highlight the importance of intra-hepatic shunting in predisposing to this syndrome. However it may also suggest that Non-cirrhotic ascites is less susceptible to infection.

The organisms that causes spontaneous bacterial peritonitis are not killed by opsonin alone but also require the presence of phagocytic cells[31].

However phagocytic cells, macrophages and PMN are ineffective in phagocytosing bacteria in the absence of specific and nonspecific opsonins such as complement, fibronectin and immunoglobulins, in patients with cirrhotic ascites compared to patients with other forms of ascites. The complement levels in the ascitic fluid are directly proportional to the levels of the protein.

The opsonic activity of the ascitic fluid too correlates closely with the protein content[9]. Part of the reason for the susceptibility of cirrhotic ascites to spontaneous bacterial peritonitis may be the low levels of specific and nonspecific opsonins required for the phagocytosis to take place successfully in the fluid.

Absence of spontaneous bacterial peritonitis in ascites not due to the nephrotic syndrome or liver disease may also be related to the higher concentration of protein in such fluids and to relative protection that this protein affords against the infection.

The most common causative organism isolated from the ascitic fluid of these patients are Gram negative bacteria especially E.coli and Klebsiella pneumoniae. Gram positive cocci are isolated less frequently. The enteric nature of most organisms that cause these infections implicates the gut as their source. However direct passage of bacteria from the intestinal lumen to ascitic fluid has not been documented, unless the gut mucosa integrity has been lost[32].

In summary, the pathogenesis of spontaneous bacterial peritonitis is due to the combination of prolonged bacteremia due to the intra-hepatic shunting and absence of effective bacterial destruction in ascites which has low protein content.

MICROBIOLOGICAL PROFILE OF SBP

Regarding the etiology, over 60% of the SBP episodes are produced by Gram-negative enteric bacilli – E.coli and Klebsiella pneumonia being the most frequently isolated microorganisms. [33, 34]

It has been ascertained that certain E. coli strains can translocate the intestinal mucosa more often – probably because of a higher capacity to adhere to it and because of a higher virulence that determines a higher resistance to the defence mechanisms of the host.

In about 25% of the cases, gram-positive cocci are involved: streptococci (frequently pneumococcus) and enterococci.[35, 36]

Although the bowel flora is predominantly anaerobic, SBP is very seldom produced by anaerobic microorganisms, due to their incapacity to translocate the intestinal mucosa and due to the high volume of oxygen in the intestinal wall and in the tissues that surround it.

A special situation is represented by the patients that receive antibiotic treatment (usually fluoroquinolones) for the inhibition of the gram-negative intestinal flora (selective intestinal decontamination), with the purpose of reducing the incidence of the SBP episode. In these patients, an increased frequency of the SBP episodes produced by gram-positive bacteria has been ascertained.[37]

Streptococci were found more frequently in community-acquired episodes (53.8%) than in nosocomial episodes (33.3%). Gram-negative bacilli were

significantly more frequent in nosocomial episodes than in community-acquired episodes.

MORTALITY / MORBIDITY

The SBP mortality rate ranges from 40-70% in adults patients with cirrhosis and is lower in children with nephrosis.

- Patients with concurrent renal insufficiency have been shown to be at a higher risk of mortality from SBP than those without concurrent renal insufficiency.
- Recent reports show that mortality from SBP may be decreasing among all subgroup of patients because of advances in its diagnosis and treatment.

Race: No race predilection is known.

Sex: In patients with ascites, both sexes are affected equally.

Age: While the etiology and incidence of hepatic failure differ between children and adults, in those individuals with ascites, the incidence of SBP is roughly equal.

PREDISPOSING FACTORS

Spontaneous bacterial peritonitis occurs only in the setting of liver disease, for all practical purposes. Specific subsets of patients with cirrhosis and ascites have been identified as having an unusually high risk for development of spontaneous bacterial peritonitis.

They are

1. Severity of the liver disease.
2. Gastro-intestinal Haemorrhage.
3. Ascitic fluid protein ≤ 1 gm/dl.
4. UTI
5. Urinary Bladder Catheters.
6. Intravascular Catheters.
7. Previous H/O spontaneous bacterial peritonitis.

Severity of liver disease is a significant risk factor for spontaneous bacterial peritonitis. 95% of patients with spontaneous bacterial peritonitis have an elevated Serum Bilirubin and 98% have prolonged prothrombin time. Cirrhotic patients with GI Haemorrhage are in fact, at the time of admission, are at risk of developing bacterial infections during the initial 3 to 4 days. Acute haemorrhage depresses the reticulo-endothelial functions, alters intestinal permeability and favours bacterial translocation, which is higher in the cirrhosis.

The antimicrobial activity of the ascitic fluid correlates

(1) Directly with the ascitic fluid protein content and also with the ascitic fluid C3 levels and

(2) Inversely with risk of spontaneous bacterial peritonitis.

The total ascitic fluid protein levels less than 1gm/dl is associated with ten fold increased risk for development of spontaneous bacterial peritonitis, compared with the patients whose ascitic fluid total protein levels are greater than 1gm/dl. Recurrence is also common with lower ascitic fluid protein levels.

UTI have been reported to be common in patients with cirrhosis. The association between spontaneous bacterial peritonitis and urinary tract infection is evident in one of the prospective studies.[38]

Invasive procedures other than endoscopy have been incriminated in predisposing to bacteremia and spontaneous bacterial peritonitis in cirrhotic patients with ascites. Intravascular and bladder catheters are portals of entry of bacteria into these immunocompromised hosts and they should be avoided whenever possible.

Previous spontaneous bacterial peritonitis episodes may predispose to recurrence. These patients, who are immunodeficient for one spontaneous bacterial peritonitis episode, are at a risk for more episodes. Repeated large volume paracentesis have been reported to deplete protein levels and they could predispose to spontaneous bacterial peritonitis. Studies which have compared diuretic therapy to paracentesis have shown that, ascitic fluid opsonic activity and **C3** levels increased in the diuretic group, whereas ascitic fluid opsonic activity remained stable and ascitic fluid **C3** levels decreased in the single large volume paracentesis plus diuretic group. No variations in serum C3 levels were observed. Diagnostic paracentesis is seldom a cause for the development of spontaneous bacterial peritonitis. Poly-microbial

bacterascites may be possible during paracentesis, where the bowel is entered by the paracentesis needle and bowel contents released into the ascitic fluid.

Bacteremia and spontaneous bacterial peritonitis have been reported after endoscopy, particularly after an emergency sclerotherapy. However, consensus is that, development of bacterial infections in bleeding cirrhotic patients treated with sclerotherapy may be related more to the GI bleeding than to the endoscopic procedure.

Hepato-cellular carcinoma may suppress the host defenses against bacterial infections. However most patients with tumour have underlying cirrhosis. Studies have shown that the development of spontaneous bacterial peritonitis depends on the severity of the underlying liver disease and not due to the presence of the tumour. [39, 40]

FACTORS NOT PREDISPOSING TO THE DEVELOPMENT OF SPONTANEOUS BACTERIAL PERITONITIS

1. Diagnostic paracentesis
2. Endoscopy
3. Sclerotherapy
4. Banding of the varices
5. Hepato-cellular carcinoma

DIAGNOSIS

Clinical signs and symptoms of peritonitis are usually mild in patients with spontaneous bacterial peritonitis, compared with patients who have surgical peritonitis in the absences of ascites. The ascitic fluid prevents the development of a surgical abdomen. Fever and abdominal pain are the most common symptoms in spontaneous bacterial peritonitis. A high proportion of patients with spontaneous bacterial peritonitis have non-specific symptoms such as hepatic encephalopathy, diarrhoea, ileus, hypotension and shock. More over, spontaneous bacterial peritonitis may manifest only as a mild deterioration in mental status or mild azotemia or acidosis. 10% to 33% of patients with spontaneous bacterial peritonitis may be asymptomatic; hence there should be a high index of suspicion to diagnose spontaneous bacterial peritonitis.

Ascitic fluid analysis is necessary for the diagnosis of spontaneous bacterial peritonitis. An ascitic fluid PMN count of 250 or more provides presumptive evidence of ascitic fluid infection. Gram stain of ascitic fluid usually demonstrates no bacteria in patients with cirrhosis who have spontaneous bacterial peritonitis, because concentration is very low (≤ 10 organisms/ml) and about 10,000ml are required before bacteria are apparent by this technique. Gram stain can be very helpful in identifying patients with gut perforation, where multiple types of bacteria are seen usually.

Acute phase reactants are produced during bacterial infections. The values of serum and ascitic fluid acute phase reactants levels in spontaneous bacterial peritonitis have been studied. TNF - **α,** Interleukin-6, **α 1 -** antitrypsin and CRP levels are higher in infected compared with sterile ascitic fluid.

Ascitic fluid should be cultured in blood culture bottles. It is important to culture an adequate volume, 10-20ml/bottle. It is also important to inoculate the bottles with ascitic fluid at the bedside. A four-hour delay in inoculating the blood culture bottles results in a 25% reduction in detection of the cultures.

BacT/Alert., is an automated calorimetric microbial detection system that provides an earlier microbiological diagnosis of bacteremia, than conventional bottles, without loss of sensitivity. The time lapsed for ascitic fluid culture positivity was 13.3 hours for BacT/Alert., and 43 hours for conventional blood culture bottles in patients with spontaneous bacterial peritonitis.

MANAGEMENT

Empirical treatment of suspected spontaneous bacterial peritonitis must be started as soon as possible.

1. Immediately after obtaining fluid for culture and analysis, when bacterial infections are clinically suspected based on signs and symptoms.

2. When the neutrophil count of ascitic fluid is ≥ 250 cells/mm^3.

Before 1895, the combination of an aminoglycoside and a Beta lactam antibiotic was the most frequently used empiric antibiotic regimen in cirrhotic patients with spontaneous bacterial peritonitis. Resolution of infection was achieved in less than 60% of patients and aminoglycoside nephrotoxicity developed in most cirrhotic patients even if aminoglycoside serum levels were below the toxic range.

In 1985, Felisart et al., demonstrated in a randomised control trial that I.V. cefotaxime was more effective and less toxic than Ampicillin-Tobramycin combination in cirrhotics with severe infections. The side effects were also negligible with cefotaxime and no one treated with cefotaxime developed drug induced ATN.[41] Similar studies worldwide showed similar experience, and aminoglycoside use was abandoned approximately 10 years ago. The half-life of cefotaxime is prolonged in patients with cirrhosis when compared with the patients without liver disease. Therefore recent studies have focused in dose reductions in cirrhotic patients with spontaneous bacterial peritonitis. [42, 43]

Treatment given with 2.0 grams of cefotaxime given I.V. every 8 to 12 hours was as effective as a 6th hourly regimen in a randomised trial.[43] The length of therapy has also been recently clarified, short course therapy (5days) has been shows to be as effective as long course therapy (10 days). [44]

Other 3rd generation cephalosporins such as ceftriaxone when given 2 grams per day cover most of the flora responsible for spontaneous bacterial peritonitis. Aztreonam and Amoxycillin plus clavulanic acid are some of the non-cephalosporin drugs that have been used in the treatment of spontaneous bacterial peritonitis. However the efficacy of Aztreonam when compared with amoxycillin plus clavulanic acid was less in the treatment of spontaneous bacterial peritonitis.

Because of the relatively good conditions of most patients with spontaneous bacterial peritonitis during early phase of infection, treatment with oral antibiotics was suggested. The oral Quinolones, which have good ascitic fluid penetration and a reasonably good spectrum of coverage, are also used.

In patients with uncomplicated SBP (no gastrointestinal bleeding, hepatic encephalopathy, ileus, shock or renal failure), treatment with Ofloxacin or other oral quinolones for 8 days can be administered.[45, 46, 47]

A good response to therapy can be evaluated by clinical criteria (disappearance of infection signs and symptoms), but the most important parameter remains the decrease to a half (from the pre-treatment value) of the PMN number in the ascitic fluid obtained by paracentesis after two days of treatment. [48]

Studies that require further confirmation propose the association of albumin infusion (1.5 g/kg body weight the first day, then 1 g/kg three more days) to the Cefotaxime treatment for patients with renal failure and SBP. Albumin in these patients may improve the renal function by increasing the intravascular volume, because vasodilatation induced by cytokines released in excess reduces the effective arterial volume.[46, 49]

Other adjuvant therapies in patients with SBP include prokinetics and probiotics.

Prokinetics are used to shorten the intestinal transit time, reducing thus the intestinal bacterial overgrowth and the risk of bacterial translocation.

Encouraging results have been obtained by using Cisapride and Propranolol, the latter's β blocking effect antagonizes the increased adrenergic tone existent in patients with cirrhosis and responsible for the decreased intestinal motility.

Probiotics are used for intestinal flora reequilibration, in favour to anaerobic protective bacteria. Bacteriotherapy with Lactobacillus seems to correct intestinal bacterial overgrowth, to stabilize mucosal barrier function and to stimulate the local defence mechanisms.[34, 50]

Oral treatment with conjugated bile acids (cholylglycine and cholylsarcosine) for preventing BT is under evalution.[34]

FLOW CHART- II

MANAGEMENT OF CIRRHOTIC PATIENTS WITH SBP

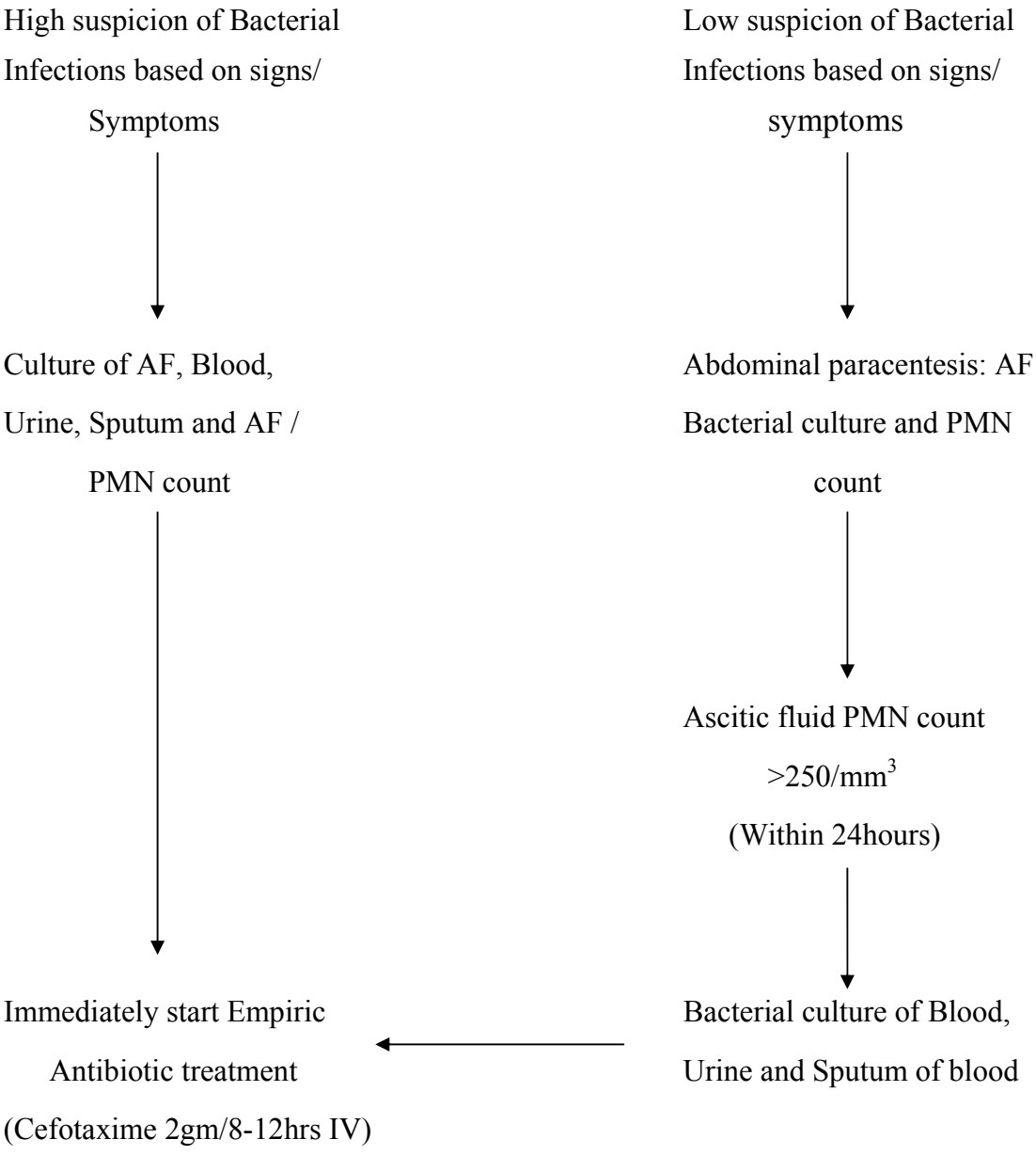


TABLE - I

RESULTS OF DIFFERENT ANTIBIOTIC TREATMENTS IN CIRRHOTIC PATIENTS WITH SBP

AUTHOR (REFERENCE)	ANTIBIOTIC	DOSAGE	NUMBER OF PATIENTS	INFECTION RESOLUTION (%)	SURVIVAL OF HOSPITALI SATION (%)
Runyon[44]	Cefotaxime	2g/ 8 hrs	43	93	67.4
Rimola[43]	Cefotaxime Cefotaxime	2g/6hrs. 2g/12hrs	66 70	77 79	69 79
Ariza[51]	Aztreonam	0.5 gm/8hrs.	16	56	63
Gomez[52]	Cefotaxime Cefonicid	2g/24hrs 2g/12hrs	30 30	100 94	70 63
Grange[53]	Amp./Clauv.	1gm/6 hrs	27	85	60
Navaeu[54]	Oral oflox. Cefotaxime	400mg/12hrs 2gm/6hrs	64 59	84.4 84.7	79.7 80.9

PROPHYLAXIS

Prevention of bacterial infection should be considered in patients with liver disorders, who are at a high risk. General measures that have been proposed to reduce the incidence of infections in cirrhotics include discontinuing alcohol intake, reducing the length of hospitalization, avoiding unnecessary instrumentation (especially bladder and IV catheters), improving the nutritional status and treating the complications of cirrhosis such as ascites formation and GI haemorrhage. Because, enteric aerobic gram-negative bacteria are the most frequently isolated bacteria in spontaneous bacterial peritonitis, selective intestinal decontamination (SID) has been proposed as a method for the prevention. SID consists of inhibition of the problematic gram-negative flora of the gut with preservation of commensal bacteria. Anaerobes comprise more than 99% of the gut flora and their presence is very important in preventing intestinal colonistation, overgrowth and subsequent extra–intestinal dissemination of pathogenic bacteria. Randomised clinical trials have demonstrated efficacy of SID in preventing gram negative bacterial infection in neutropenic patients without overgrowth of resistant bacteria or significant side effect.

There are three categories of cirrhotic patients which are more at risk of development SBP. Patients with

(1) GI hemorrhage

(2) Ascitic fluid protein level < 1 g/dl

(3) Previous history of SBP episode.

For preventing SBP in patients with low ascitic fluid protein level, Norfloxacin is administrated during hospitalization.

Patients with GI hemorrhage are more at a risk in developing SBP; it is considered that 20% of them have SBP at admission and 30-40% will develop an infection during hospitalization. These patients will receive 800 mg/day Norfloxacin through the nasogastric tube for 7 days.[47, 50]

In patients who survive an episode of SBP, a long-term prophylactic treatment (for preventing recurrence) with Norfloxacin 400 mg/day will be administered.

When the oral administration or the administration by nasogastric tube Norfloxacin is not possible, Ciprofloxacin can be administered i.v. In patients with intolerance to quinolones, the combination of trimethoprim / sulfamethoxazole for 5 days / week can be used.

Patients who receive primary or secondary prophylactic treatment with Norfloxacin can develop resistant gram-negative bacilli strains. Fortunately, no crossed resistance between third generation cephalosporins and quinolones has been observed, thus infections caused by quinolone resistant germs can be treated with Cefotaxime or Ceftriaxone.

Other prophylactic measures include:

- Diuretics, which reduce the ascites volume and increase the ascitic fluid opsonic activity;

- Local infections treatment and eradication, before their disseminations;

- Porto-caval shunts and TIPS (transjugular intrahepatic portosystemic shunt) for GI hemorrhage or ascites risk reduction, reducing indirectly SBP risk;
- Abstinence from alcohol in case of alcoholic cirrhosis.[60]

CURRENT INDICATIONS FOR SID IN CIRRHOTIC PATIENTS

TABLE – II

GROUP	DOSE OF NORFLOXACIN
Cirrhotic patients with GI Haemorrhage	400 mg p.o.bd x7 days
Cirrhotic patients with Ascitic fluid protein < 1gm/dl	400 mg/day p.o. during the entire stay
Cirrhotic patients who have survived an episode of SBP	400 mg/day indefinitely or until liver transplantation

Spontaneous bacterial peritonitis is a maker for advanced liver disease and poor survival. Patients who survive an episode should be considered for liver transplantation. Many patients who are awaiting liver transplantation in the west are receiving quinolone prophylaxis.

MATERIALS AND METHODS

This prospective study includes 75 patients with cirrhosis of liver with ascites diagnosed on the basis of clinical evaluation, biochemical investigations and ultrasonography admitted to the medical wards of the Thanjavur Medical College Hospital, Thanjavur during the period October 2005 to September 2007.

The **inclusion criteria** were

a) Patients with clinical features of cirrhosis with ascites.

b) Ultrasonographic evidence of cirrhosis.

c) Those who had not been started on antibiotics, before admission.

The **exclusion criteria** were

Patients with

a) Non-cirrhotic portal fibrosis

b) Cholelithiasis

c) Hydatid cysts in the liver

d) Secondaries in the liver

e) Amoebic liver abscess

f) Ascites due to renal, tubercular or malignant pathology.

Liver cirrhosis was **diagnosed** on the basis of

a) **Biochemical** abnormalities like serum Bilirubin, serum albumin, SGOT, SGPT.

b) **Ultrasonography** showing shrunken or enlarged liver, nodular surface, increased echotexture, portal vein dilatation.

c) **Ascitic fluid** study.

There were **75** patients of whom **56** were **males** and **19** were **females**. The age group was between **27** to **65**years. **Mean age** was **45.12** years. The Proforma needed for the study took into account of the presenting clinical features of the patients on admission, the routine haemotological, bio-chemical investigations, ascitic fluid analysis and liver function tests. The ascitic fluid was analysed with utmost importance.

ASCITIC FLUID ANALYSIS

The samples were collected prior to the administration of antibiotics to the patients. The skin was disinfected with povidone iodine solution. The hands were scrubbed with soap and sterile disposable gloves were used to reduce the contamination of the skin. As most patients had tense ascites, the technique of a 'Z' tract, which minimises leakage, was used. About 10ml of the ascitic fluid was inoculated into the blood culture bottles at the bedside. This methods was followed to get a higher yield as the delay in the inoculation reduced the detection of positive culture by 25%.[51] The bottles were incubated for 72 hours and those of which showed

growth were plated for identification. Another 5ml was sent to the microbiological laboratory for culture by the conventional technique using agar plates as done routinely. About 1ml of the fluid was sent for cell count and in all cases cell count was done as soon as possible. The remainder of the fluid was sent for bio-chemical analysis. The results were tabulated in the master chart to facilitate analysis.

SBP was suspected clinically in patients presenting with fever, abdominal pain, tenderness and further confirmation was done by ascitic fluid cell count and culture.

Statistical evaluation was performed and analysis done.

RESULTS

In this study, out of **75** cirrhotic patients with ascites, the number of **males** were **56** (74.67%) while the number of **females** were **19** (**25.33%).** The age groups ranged from **27** to **65** years. The mean age was **45.12** years.

TABLE III

SEX	NO. OF PATIENTS IN THE STUDY	PERCENTAGE OF PATIENTS IN THE STUDY
MALE	56	74.67%
FEMALE	19	25.33%

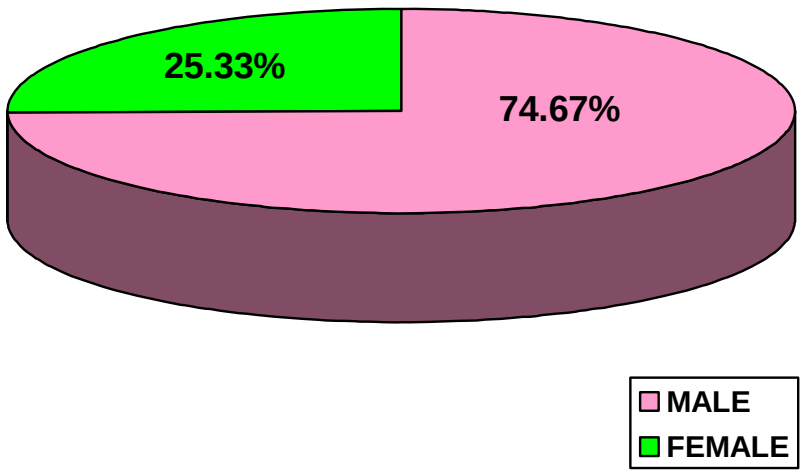


FIG.1

PREVALENCE

In this study, **19** out of **75** patients had spontaneous bacterial peritonitis, which means that **25.33%** of cirrhotic patients with ascites had spontaneous bacterial peritonitis, on admission. Hence the prevalence in this hospital was **25.33%**.

TABLE IV

NO. OF PATIENTS IN THE STUDY	NO. OF PATIENTS WITH SBP	PREVALENCE
75	19	25.33%

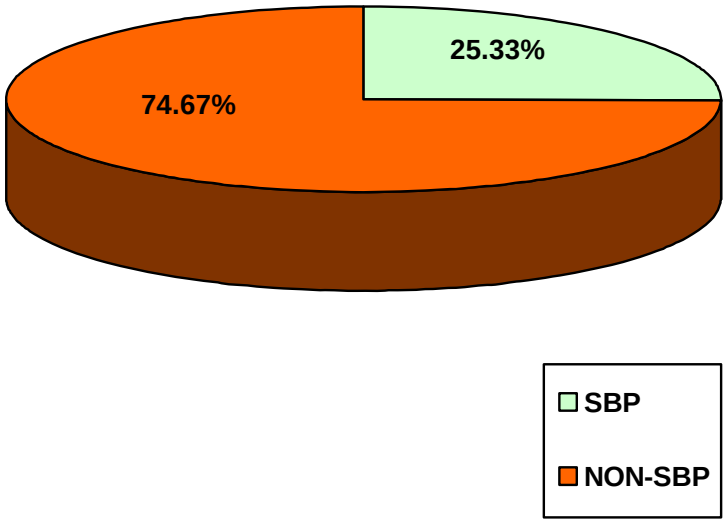


FIG. 2

AGE WISE DISTRIBUTION OF SBP IN CIRRHOTIC PATIENTS

In this study**, 68.42% (13** out of **19)** of patients with spontaneous bacterial peritonitis were in the age group of **41** to **50** years. **15.79 %(3** out of **19)** of patients with spontaneous bacterial peritonitis were in the age group of **51** to **60 years.** There were **2** patients in the age group of **31** to **40** years, constituting **10.53%** and 1 patient in the age group of **61** to **70** years**,** constituting **5.26%.**

TABLE – V

AGE IN YEARS (group)	NO.OF PATIENTS WITH SBP	PERCENTAGE
0-10	-	-
11-20	-	-
21-30	-	-
31-40	2	10.53
41-50	13	68.42
51-60	3	15.79
61-70	1	5.26

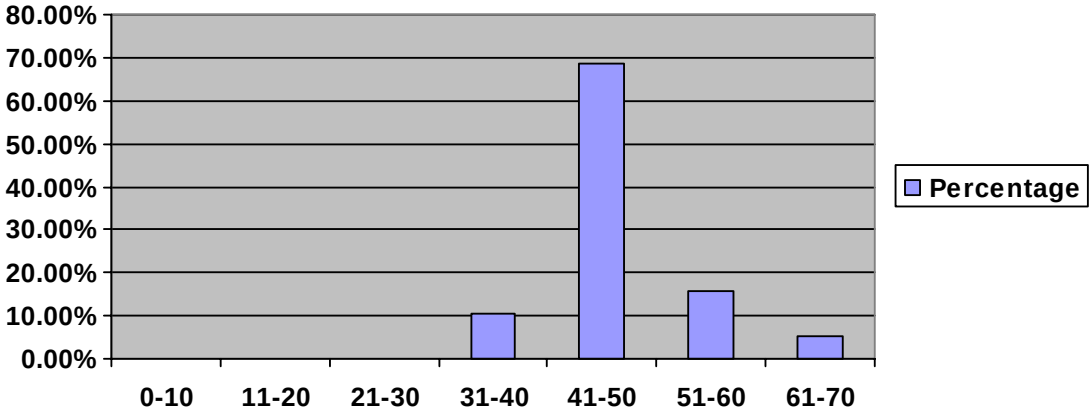


FIG 3

SEX DISTRIBUTION

In this study, 25% (14 out of 56) of males and 26.32% (5 out of 19) of female patients had SBP.

TABLE VI

SEX	NO. OF PATIENTS IN THE STUDY	NO. OF PATIENTS WITH SBP	PERCENTAGE
MALE	56	14	25%
FEMALE	19	5	26.32%

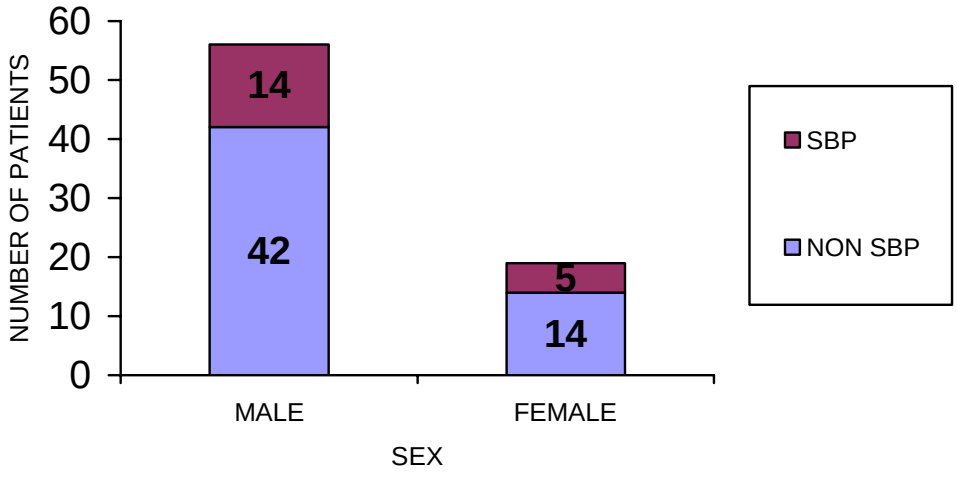


FIG 4

CLINICAL FEATURES IN CIRRHOTIC PATIENTS WITH SBP

TABLE – VII

CLINICAL FEATURE	NO. OF PATIENTS	PERCENTAGE
ABDOMINAL PAIN	16	84.21
TENDERNESS	11	57.89
FEVER	10	52.63
GI BLEED	6	31.59
HEPATIC ENCEPHALOPATHY	2	10.53
RENAL FAILURE	2	10.53

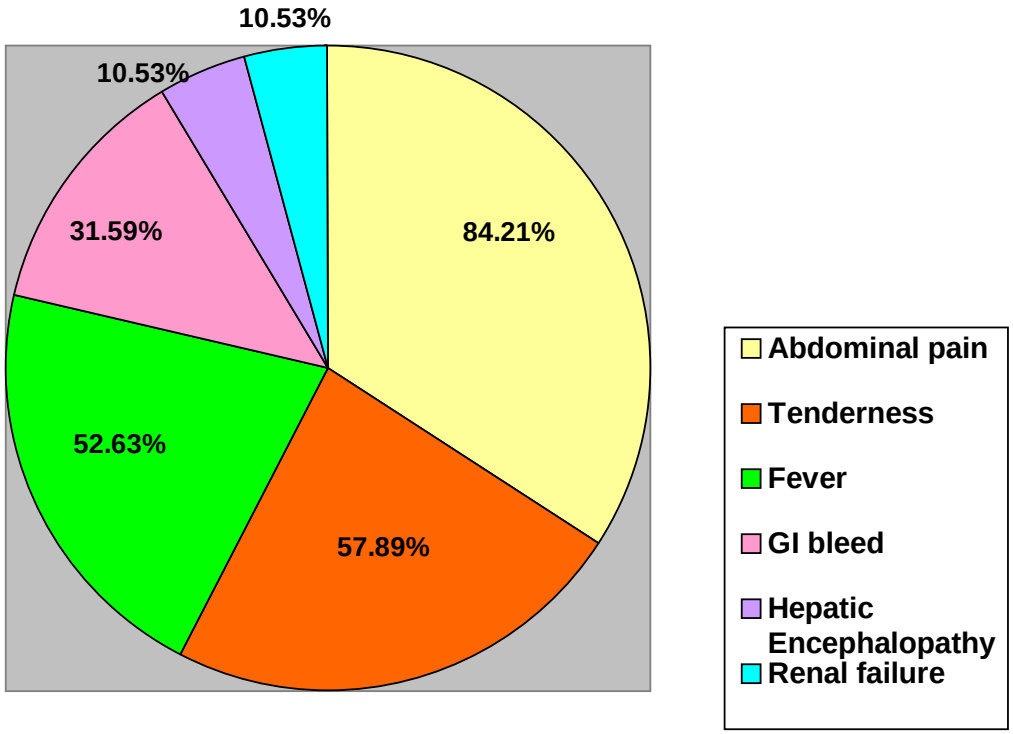


FIG. 5

Abdominal pain constituted the major symptom in **84.21%** of patients while abdominal tenderness was the next commonest feature occurring in **57.89%** of cases. Fever occurred in **52.63%** of cases. GI bleed occured in **31.58%** of cases, followed by hepatic encephalopathy and renal failure in **10.52%** of cases each.

CLINICAL FEATURES IN CIRRHOTIC PATIENTS WITHOUT SBP

TABLE – VIII

CLINICAL FEATURE	NO. OF PATIENTS	PERCENTAGE
ABDOMINAL PAIN	19	33.93
TENDERNESS	3	5.36
FEVER	13	23.21
GI BLEED	3	5.36
HEPATIC ENCEPHALOPATHY	2	3.57
RENAL FAILURE	2	3.57

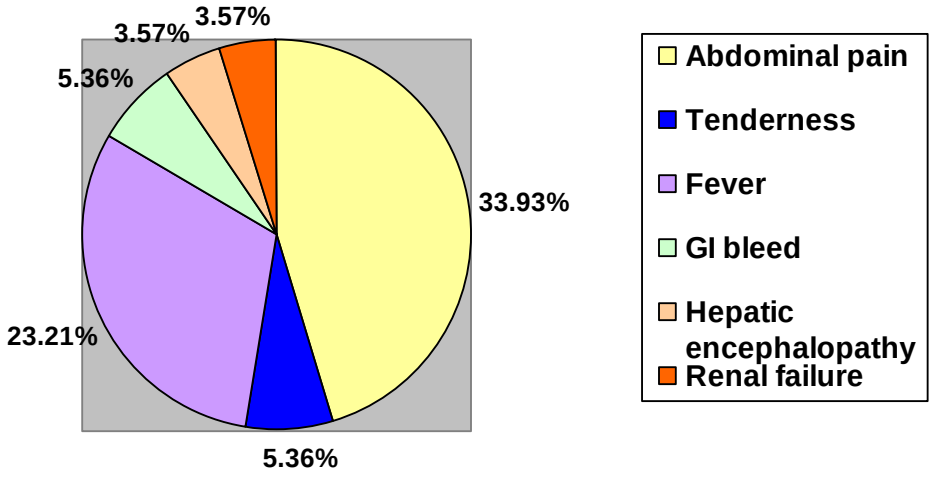


FIG. 6

ASCITIC FLUID BIOCHEMISTRY

In this study, **16** out of **19** patients (84.21%) with spontaneous bacterial peritonitis had ascitic fluid protein level less than **1** gm/dl. The mean value of ascitic fluid protein in patients with spontaneous bacterial peritonitis was 0.88 gm/dl. The mean value of ascitic fluid protein in Non SBP patients was **1.1 gm/dL**

TABLE IX

ASCITIC FLUID PARAMETER	MEAN VALUE IN SBP PATIENTS	MEAN VALUE IN NON SBP PATIENTS
PROTEIN	0.88 gm/dl	1.1 gm/dl

SEVERITY OF LIVER DISEASE

The serum bilirubin was elevated in all patients with spontaneous bacterial peritonitis, with a mean value of **2.66%** mg/dl. The PT was prolonged by at least 2 times than that of control, in **14** out of **19** patients, that is 73.68% of patients with spontaneous bacterial peritonitis. The serum albumin was reduced in all SBP patients with a mean value of **3.03** gm/dl.

TABLE X

LIVER PARAMETER	MEAN VALUE IN SBP PATIENTS
SERUM BILIRUBIN	2.66mg/dl
SERUM ALBUMIN	3.03gm/dl
PROTHROMBIN TIME	2 X CONTROL

CHILD'S CRITERIA FOR GRADING THE SEVERITY OF LIVER DISEASE

In this study, out of 19 patients with SBP, 14 patients **(73.68**%) were in child's class C, 4 patients **(21.05%)** were in child's class B and 1 patient **(5.26%)** was in child's class A.

TABLE XI

CHILD'S CLASS	NO. OF PATIENTS WITH SBP	PERCENTAGE
A	1	5.26%
B	4	21.05%
C	14	73.68%

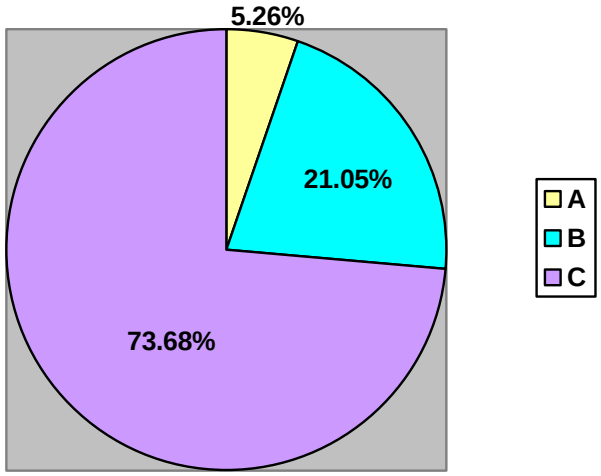


FIG 7

ASCITIC FLUID CULTURE

Ascitic fluid cultures using blood culture bottles detected growth in **all** cases of spontaneous bacterial peritonitis where as conventional methods could do so only in **47.37%** of cases

TABLE XII

METHOD	NO. OF PATIENTS WITH SBP YIELDED GROWTH	PERCENTAGE OF DETECTION
BLOOD CULTURE BOTTLE	19	100%
CONVENTIONAL	9	47.37%

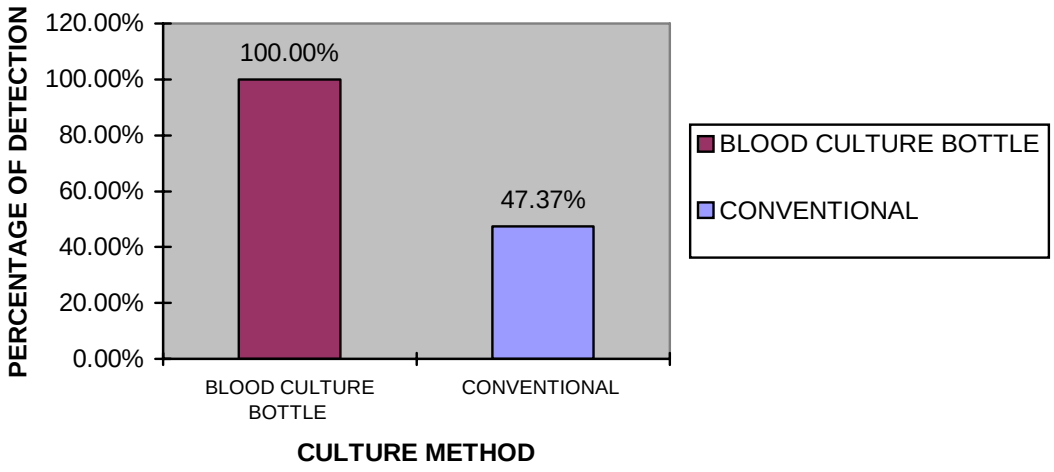


FIG 8

VARIANTS OF ASCITIC FLUID INFECTIONS

More over in this study, out of the 75 cirrhotic patients with ascites **10 (10.33%)** patients had positive culture for one organism, but the neutrophil cell count was less than 250 cells/mm3. This group of patients had mono-microbial nonneutrocytic ascites.

TABLE XIII

Sl.No	VARIANTS OF A.F.I	NO. OF PATIENTS	PERCENTAGE
1	Spontaneous Bacterial Peritonitis	19	25.33%
2	Mono-microbial non neutrocytic ascites	10	10.33%
3	Culture negative neutrocytic ascites	0	0
4	Secondary bacterial peritonitis	0	0
5	Poly-microbial bacterascites.	0	0

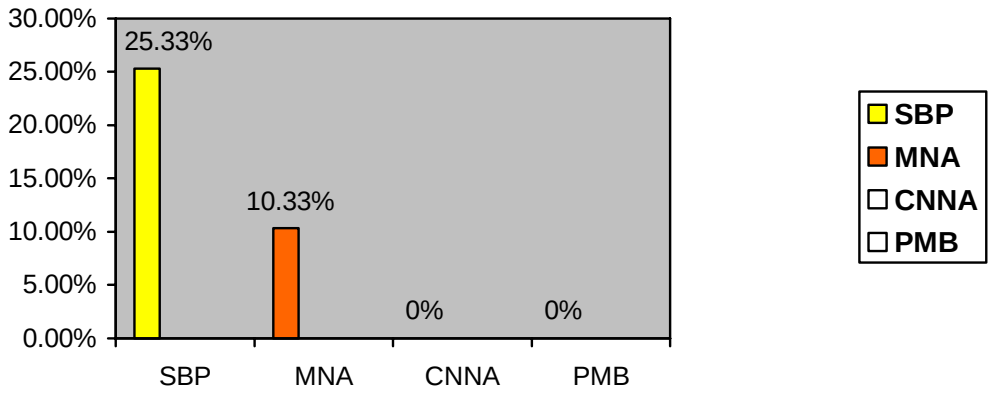


FIG 9

MICROBIOLOGICAL PROFILE

TABLE XIV

ORGANISM	NO. OF PATIENTS	PERCENTAGE
E.COLI	8	42.11%
KLEBSIELLA PNEUMONIAE	7	36.84%
PROTEUS	2	10.53%
PSEUDOMONAS	1	5.27%
STAPHYLOCOCCUS AUREUS	1	5.27%

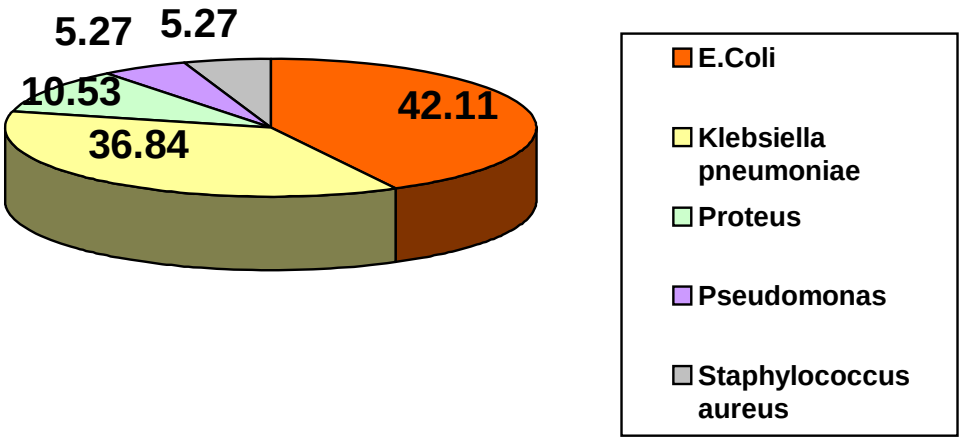


FIG 10

E.coli was the most common pathogen isolated in **42.11%** (**8** out of **19**) of the patients with spontaneous bacterial peritonitis, while **Klebsiella pneumoniae** was isolated in **36.84%** (**7** out of **19**) of the patients followed by **Proteus** in two patients, that is **10.53%, Pseudomonas** in one patient, that is **5.27%** and **Staphylococcus aureus** in one patient, that is **5.27%.**

DISCUSSION

PREVALENCE

In this study, the prevalence of spontaneous bacterial peritonitis in cirrhotics with ascites was **25.33%** (**19** out of **75** patients). **TABLE IV**

In various studies world wide, the cumulative probability of spontaneous bacterial peritonitis occuring during hospitalization in a patient with cirrhosis has been reported to vary from **8 % to 27 %** [3, 4].

. In the present study it was seen in **25.33 %** of the patients. Bacterial colonisation of ascitic fluid is related to its markedly deficient bacterial and opsonic activity in cirrhosis. In this context, alcoholic and non-alcoholic cirrhosis behaves in a similar fashion.

AGE AND SEX DISTRIBUTION

Among **75** patients with cirrhosis and ascites admitted to this hospital **74.67 %** were **male**s where as females constituted only **25.33 %**. This could be explained by the fact that, a large group of cirrhotic patients in our part is due to alcoholism and female alcoholics are fewer in numbers.

The prevalence of spontaneous bacterial peritonitis was maximum in the age group of **41** to **50** years, which is **73.69%**. Out of which, **84.62%** (**11**out of **13** patients) were **males** and **15.38%** (**2** out of **13** patients) were **females**. **TABLE V**

Studies have shown that incidence of spontaneous bacterial peritonitis is equal in all age groups. However, in this study prevalence was maximum in the age group of **41** to **50 years**. This could be explained by the fact that mean age in this study was **45.12 years**.

In this study, **25% (14 out of 56)** of males and **26.32% (5 out of 19)** of female patients had SBP. **TABLE VI**

Studies have shown that both sexes are affected equally with spontaneous bacterial peritonitis in patients with ascites.

CLINICAL FEATURES

The main presenting feature was **abdominal pain** seen in **84.21%** (**16** out of **19** patients) of patients with spontaneous bacterial peritonitis, while **abdominal tenderness** was present in **57.89%** (**11**out of **19** patients) of patients. **Fever** was a presenting feature in **52.63%** (**10** out of **19** patients) of patients with spontaneous bacterial peritonitis, while only **23.21%** (**13** out of **56** patients) of patients without spontaneous bacterial peritonitis had fever. **TABLE VII**

In the study by **N.Venkataraman, N.Kasirajan et al.,** vague abdominal pain was the only symptom present in the patients with spontaneous bacterial peritonitis.

.

Upper GI bleed was present in **31.58%** (**6** out of **19** patients) of patients who were diagnosed as spontaneous bacterial peritonitis. **Bleichner G et al.,** showed that

more than **20 %** of cirrhotics with GI bleed are infected at the time of admission. **Rimola et al.,** and **Soriano et al.,** also showed that the bacterial infection develops in an additional **30%** of those with upper GI bleed during hospitalisation usually during the first **3 to 4** days. [31, 43]

Hepatic encephalopathy and renal failure was seen in **10.53%** (**2** out of **19** patients) of patients with spontaneous bacterial peritonitis at presentation.

SEVERITY OF LIVER DISEASE

The severity of liver disease is a major risk factor for the development of spontaneous bacterial peritonitis.[3, 39, 55] Severity is assessed by increased prothrombin time and increased serum bilirubin levels.

In this study, serum bilirubin was elevated in all patients with spontaneous bacterial peritonitis, with a mean level of **2.66%** mg/dl.

In the study by **Cirera I ,Bauer TM ,Navasa M et al,** [19] serum bilirubin level **>2.5 mg/dl** is an independent predictive factor of SBP. In this study mean level of serum bilirubin in SBP patients was **2.66 mg/dl**.

The PT was prolonged by at least 2 times than that of control, in **73.68% (14 out of 19)** of patients with spontaneous bacterial peritonitis.

In the Study by **Guarner, C., Runyon BA,** [56] **95%** of patients with spontaneous bacterial peritonitis can have increased serum bilirubin levels and **98%**

of patients can have abnormal PT. In this study, serum bilirubin was elevated in all patients and PT was abnormal in **73.68%** of patients

In this study, out of 19 patients with SBP, **14** patients **(73.68%)** were in child's class **C**, **4** patients **(21.05%)** were in child's class **B** and **1** patient **(5.26%)** was in child's class **A**.

Studies have shown that **70%** of patients who develop SBP were in child C class. In this study **73.68%** of patients were in child C class.

ASCITIC FLUID ANALYSIS

In this study, **16** out of **19** patients **(84.21%)** with spontaneous bacterial peritonitis had ascitic fluid protein level **less than 1 gm/dl**. The mean value of ascitic fluid protein in patients with spontaneous bacterial peritonitis was **0.88 gm/dl**. The mean value of ascitic fluid protein in Non SBP patients was **1.1 gm/dL**

Runyon and associates have shown that cirrhotic patients with ascitic fluid protein levels of 1 gm/dl or less had ten fold increased risk for the development of spontaneous bacterial peritonitis, when compared to cirrhotic patients with ascitic fluid levels greater than 1 gm/dl.[51] This clearly shows that a low ascitic fluid proteins level is a predisposing factor for spontaneous bacterial peritonitis.

ASCITIC FLUID CULTURE

The blood culture bottles detected growth in all cases of spontaneous bacterial peritonitis**,** while the conventional methods could do so only in **47.37% of cases [9 out of 19]**, implying the superiority of the inoculation method, TABLE XII .Similar experience has been of **Pawar et al.,** too.[58]

A study by **Runyon BA, HOEFS JC,** [6] has shown that **35%** to **58%** of patients with spontaneous bacterial peritonitis are culture negative by conventional methods. In this study **47.37%** of patients with spontaneous bacterial peritonitis are culture negative by conventional method.

Multiple studies have demonstrated superior sensitivity in using blood culture bottles for culture of ascitic fluid compared with the conventional techniques.[59,60,61] Further **Runyon BA, Antillon MR** and other [62] have shown that bedside inoculation of ascitic fluid is superior to the delayed laboratory inoculation of blood culture bottles with ascitic fluid.

MICROBIOLOGICAL PROFILE

E.coli was the most common pathogen isolated in **42.11%** (**8** out of **19**) of the patients with spontaneous bacterial peritonitis, while **Klebsiella pneumoniae** was isolated in **36.84%** (**7** out of **19**) of the patients followed by **Proteus** in two patients, that is **10.53%, Pseudomonas** in one patients, that is **5.27%** and **Staphylococcus aureus** in one patient, that is **5.27%.TABLE IV**

Studies by **Runyon BA** and **Wiest R Garcia-TsaoG** [33,34] have shown more than **60%** of SBP episodes are produced by Gram-negative enteric bacilli – **E.coli** and

Klebsiella pneumonia being the most frequently isolated organisms**.** In this study **E.coli** and **Klebsiella pneumonia** were isolated in **78.95%** of SBP patients.

The commonest organism was **E.coli** followed by **Klebsiella pneumoniae,** like **Monserrat et al.,** [63] who also isolated enteric organisms, in his study.

CONCLUSION

1. Prevalence of Spontaneous Bacterial Peritonitis in this study was **25.33%.**

2. The most common presenting feature of SBP was **abdominal pain** followed by **abdominal tenderness** and **fever**. Since a large percentage of patients had abdominal pain, one must have high index of suspicion of SBP and should do cytology and culture of the ascitic fluid, more so, when the patient is febrile and abdominal tenderness is present.

3. **Low ascitic fluid protein** is a **predisposing factor** for Spontaneous Bacterial Peritonitis.

4. **Severe Liver cell dysfunction** as evidenced by the **increased serum bilirubin** and **prolonged prothrombin time** has been associated with the increased incidence of Spontaneous Bacterial Peritonitis.

5. Spontaneous Bacterial Peritonitis is a **common complication in Child's class B** and **C** cirrhotic patients**.**

6. Ascitic fluid culture using the **blood culture bottles with bedside inoculation is superior to the conventional routine culture methods** and it should be done in all suspected cases of Spontaneous Bacterial Peritonitis.

7. The **commonest organism isolated was E.coli** followed by K.pneumoniae.

8. Early recognition and treatment of SBP could reduce the morbidity and mortality of patients with cirrhosis liver and ascites and improve their quality of life.

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PROFORMA

SPONTANEOUS BACTERIAL PERITONITIS IN CIRRHOSIS LIVER WITH ASCITES

NAME **AGE** **SEX: M / F.** **IP NO:** **WARD**

COMPLAINTS **DURATION**

Fever

Pain Abdomen

Jaundice

UGI bleed

Oliguria

Altered Sensorium

PERSONAL HISTROY

Alcoholism : Y / N

PHYSICAL FINDINGS

Nutrition Pulse:

Icterus BP:

Flapping Tremor Temp:

EXAMINATION OF ABDOMEN

TENDERNESS

FREE FLUID

ORGANOMEGALY

INVESTIGATIONS

BLOOD

SUGAR UREA CREAT. ELECT. Na K H_{co3}

PROTHROMBIN TIME

URINE

ALB SUGAR DEPOSITS C & S

USG ABDOMEN PORTAL VEIN ASCITES

ASCITIC FLUID ANALYSIS PH. PROTEIN CELL COUNT

ASCITIC FLUID CULTURE

BLOOD CULTURE

CONVENTIONAL

LIVER FUNCTION TEST

SGOT	SGPT	BILIRUBIN	DIRECT	INDIRECT	TOTAL PROTEIN	ALBUMIN	GLOBUMIN	RATIO

CHILD'S CRITERIA FOR GRADING THE SEVERITY OF LIVER

DISEASE

CRITERIA	A (minimal)	B (moderate)	C (advanced)
S.bil.(mg/dl)	< 2	2-3	>3
S.alb. (g/dl)	>3.5	3.0-3.5	< 3.0
Ascites	None	Easily controlled	Poorly controlled
Neurologic disorder	None	minimal	Advanced
Nutrition	Excellent	good	Poor

ABBREVIATIONS

SBP - Spontaneous Bacterial Peritonitis

PMN - Polymorphonuclear

AF - Ascitic Fluid

AFI - Ascitic Fluid Infection

CNNA- Culture Negative Neutrocytic Ascites

BT - Bacterial Translocation

NO - Nitric Oxide

GI - Gastro Intestine

SID - Selective Intestinal Decontamination

MNB - Mono Microbial Non-Neutrocytic Ascites

S. No	Name	A/S	IP.NO	Fever	Pain Ab.	Tend	UGI Bl.	Jaundice	Ascites	Oliguria	Altered sensorium	Asterexis	Nutrition	S.Alb. G/dl	S.Bil. mg/dl	Bl. Urea	S. Cr.	PT	Child's class	ASCITIC FLUID CULTURE				
																				Protein mg/Dl	PMN Cell count /mm^3	Convent ional	Bed side	Organism
1	Lakhsmanan	60/M	855024	+	+	-	+	+	+	+	+	+	Poor	28	5	54	2.8	+	C	0.9	320	-	+	E coli
2	Samiammal	42/F	889086	-	-	-	-	-	+	-	-	-	Excel	3.1	1	36	0.8	-	A	1.2	100	-	-	-
3	Kaveri	45/F	873185	+	-	-	-	-	+	-	-	-	Excel	3	0.8	37	0.8	-	A	1.4	80	-	-	-
4	Elangovan	45/M	886274	+	+	-	-	-	+	-	-	-	Poor	3.2	2.1	40	0.9	-	C	0.8	340	+	+	Klebsiella pneumoniae
5	Kumaraguru	32/M	908529	-	-	-	-	-	+	-	-	-	Excel	3.1	1	35	0.8	-	A	1.3	110	-		-
6	Parthasarathy	65/M	867800	+	-	-	-	-	+	-	-	-	Poor	3.3	2	38	0.8	-	C	0.9	270	+	+	Klebsiella pneumoniae
7	Ramachandran	42/M	922849	-	-	-	-	-	+	-	-	-	Excel	3.2	1.1	36	0.9	-	A	1.1	140	-	-	-
8	Selvam	45/M	838399	+	+	-	-	-	+	-	-	--	Excel	3.1	1	38	0.9	-	A	1.5	140	-	-	Proteus
9	Vembu	40/M	876558	-	-	-	-	-	+	-	-	-	Excel	3.2	2	32	0.8	-	A	1.2	110	-	-	-
10	Chellakkannan	55/M	827472	-	+	-	-	-	+	-	-	-	Poor	3	2.2	39	0.9	+	C	0.9	280	+	+	Pseudomonas
11	Ulaganathan	45/M	839898	+	+	-	+	+	+	-	-	-	Poor	3	2	45	1.6	+	C	1.2	300	+	+	Proteus
12	Chinnaponnu	40/F	828574	-	-	-	-	-	+	-	-	-	Poor	3.1	2.1	35	0.8	-	C	1.4	260	+	+	Klebsiella pneumoniae
13	Ayyakkannu	48/M	836108	-	+	-	-	-	+	+	-	-	Good	3.2	2.5	52	2.2	+	B	1.3	270	+	+	Klebsiella pneumoniae
14	Kulanthai Therass	41/F	877726	+	+	-	--	-	+	-	-	-	Excel	3.2	1	35	0.8	-	A	1.1	90	-	-	-
15	Murugnandam	43/M	875559	-	+	-	-	-	+	-	-	-	Excel	2.9	1.1	37	0.9	-	A	1	80	-	-	-
16	Balumani	43/M	889159	+	+	-	-	+	+	-	-	-	Poor	3.2	2.5	41	1.1	+	C	0.8	300	-	+	E coli
17	Selvam	42/M	828605	+	+	-	-	-	+	-	-	-	Excel	3.3	2.8	39	0.8	-	A	0.9	110	-	-	Klebsiella pneumoniae
18	Subramani	47/M	900584	+	+	-	-	-	+	-	-	-	Poor	3.1	3	40	0.9	+	C	0.95	340	-	+	E coli
19	Dhanalakshmi	45/F	843359	+	+	-	+	+	+	+	+	+	Poor	2.2	6.5	65	3.2	+	C	0.7	460	+	+	Klebsiella pneumoniae
20	Noorjahan	42/F	840354	+	+	-	-	-	+	-	-	-	Poor	3.2	2	36	0.8	-	C	0.9	300	+	+	Proteus

21	Murugaiyan	40/m	838446	-	-	-	-	-	+	-	-	-	Poor	3.2	2.8	35	0.8	-	C	0.9	80	+	+	Klebsiella pneumoniae
22	Ganapathy	65/M	955289	-	-	-	-	-	+	-	-	-	Excel	3.1	0.6	38	0.9	-	A	0.95	70	-	-	-
23	Mohanraj	30/M	869355	-	-	-	+	+	+	-	-	-	Good	3	2	38	0.8	-	B	0.8	310	-	+	E coli
24	Parimala	40/F	845381	+	-	-	-	-	+	-	-	-	Excel	3	0.7	36	0.9	-	A	0.9	160	-	-	Klebsiella pneumoniae
25	Rajendran	35/M	909517	+	+	-	-	-	+	-	-	-	Excel	2.9	0.6	35	0.8	-	A	0.8	50	-	-	-
26	George	41/M	867500	-	+	-	-	-	+	-	-	-	Good	3.1	2.1	35	0.8	-	B	0.85	70	-	+	**Staphylococcus aureus**
27	Selvaraj	43/M	846980	-	-	-	-	-	+	--	-	-	Excel	3.1	0.5	36	0.7	-	A	0.9	90	-	-	-
28	Raju	42/M	825864	-	+	-	-	-	+	-	-	-	Good	3.2	1	38	0.9	-	B	0.5	80	-	-	-
29	Rengaraj	45/M	825551	+	+	-	+	+	+	-	-	-	Poor	3	2.5	46	1.2	+	C	0.4	310	-	+	E coli
30	Lakhsmanan	41/M	844278	-	+	-	-	+	+	-	-	-	Good	3.2	2.1	35	0.9	-	B	0.95	260	-	+	E coli
31	Marimuthu	46/M	827832	+	+	-	+	-	+	+	+	+	Poor	2.5	4.9	58	2.9	+	C	0.7	110	-	-	E.coli
32	Chellappan	43/F	869589	-	-	-	-	+	+	-	-	-	Excel	2.8	0.8	31	0.8	-	A	1.1	110	-	-	-
33	Karuppusamy	45/M	828725	-	-	-	-	-	+	-	-	-	Excel	2.7	0.9	35	0.8	-	A	0.8	130	-	-	-
34	Natarajan	35/M	908749	-	+	-	-	-	+	-	-	-	Excel	3	0.7	36	0.6	-	A	0.9	100	-	-	-
35	Kali	36/M	922180	-	-	-	-	-	+	-	+	-	Excel	3.1	1	38	0.7	-	A	1.1	110	-	-	-
36	Pondy	42/M	867582	-	-	-	-	--	+	-	-	-	Excel	3	1	32	0.9	-	A	1	90	-	-	-
37	Muthusamy	49/M	879593	+	+	-	+	+	+	+	-	+	Poor	2.4	5.2	68	3.2	+	C	0.6	140	-	-	E.coli
38	Veeran	50/M	872527	-	-	-	--	-	+	-	-	-	Excel	3.1	0.8	32	0.6	-	A	0.9	110	-	-	-
39	Kaliaperumal	52/M	900835	-	-	-	-	-	+	-	-	-	Excel	3	0.7	35	0.8	-	A	0.8	100	-	-	-
40	Tamilselvi	47/F	865359	-	+	-	-	-	+	-	-	-	Excel	3.1	0.7	38	0.9	-	A	0.7	80	-	-	-
41	Kuppusamy	48/M	958438	-	-	-	-	-	+	-	-	-	Excel	2.8	0.6	39	0.7	-	A	0.9	20	-	-	-
42	Samaiyan	55/M	859037	-	-	-	-	-	+	-	-	-	Excel	2.6	0.8	36	0.6	-	A	0.8	200	-	-	E.coli
43	Sudhaker	50/M	891737	+	+	-	+	+	+	-	-	-	Good	3.5	2.7	55	1.9	+	B	0.7	100	-	-	-
44	Ramya	48/M	920547	-	-	-	-	-	+	-	-	-	Excel	2.7	0.8	37	0.8	-	A	0.9	150	-	-	-

45	Thavamani	55/F	957324	-	-	-	-	-	+	-	-	-	Excel	2.6	0.4	38	0.9	-	A	0.9	90	-	-	-
46	Rajamanickam	41/M	870499	+	+	-	-	-	+	-	-	-	Excel	3	2.4	36	0.8	+	A	0.5	290	-	+	E coli
47	Sivakumar	43/M	930377	+	+	-	-	-	+	--	-	-	Excel	3.1	1.1	35	1.1	-	A	0.8	130	-	-	Klebsiella pneumoniae
48	Radhika	40/F	878335	+	+	-	-	-	+	-	-	-	Excel	3.1	1	37	0.8	-	A	0.9	100	-	-	-
49	Anbu	48/M	846983	-	-	-	-	-	+	-	-	-	Excel	3.2	0.8	31	0.6	-	A	0.7	130	-	-	-
50	Raju	49/M	865881	-	-	-	-	-	+	-	-	-	Excel	3.3	0.7	30	0.9	-	A	0.9	80	-	-	-
51	Subramanian	42/M	866192	-	-	-	-	-	+	-	-	-	Excel	3.2	0.5	35	0.7	-	A	0.6	50	-	-	-
52	Saminathan	53/M	879311	+	+	-	-	-	+	-	-	-	Excel	3.0	0.9	33	0.8	-	A	0.8	100	-	-	E.coli
53	Senbagam	58/F	873241	+	+	-	-	-	+	-	-	-	Poor	3.2	2.1	37	0.9	-	C	0.8	280	+	+	E coli
54	Andiappan	52/M	869984	+	-	-	-	-	+	-	-	-	Excel	3.1	0.7	30	0.8	-	A	0.7	70	-	-	-
55	Maheswaran	46/M	840238	+	+	-	+	+	+	+	-	-	Poor	3.5	2.2	52	2.2	+	C	0.8	410	-	+	Klebsiella pneumoniae
56	Samiappan	50/M	870148	-	-	-	-	-	+	-	-	-	Excel	3.2	0.5	28	0.8	-	A	0.6	90	-	-	-
57	Anusuya	60/F	958175	-	-	-	-	-	+	-	-	-	Excel	3.3	0.8	29	0.8	-	A	0.9	100	-	-	-
58	Indira	35/F	917028	-	+	-	-	-	+	-	-	-	Good	3.1	0.6	31	0.7	-	B	0.8	70	-	-	-

59	Manavalan	46/F	868179	-	-	-	-	-	+	-	-	-	Excel	3.3	0.9	35	0.8	-	A	0.6	180	-	-	E.coli
60	Tholappan	49/M	907835	-	-	-	-	-	+	-	-	-	Excel	3.2	1	30	0.9	-	A	0.8	80	-	-	-
61	Sethuraman	60/M	842433	+	+	-	-	-	+	-	-	-	Excel	3	1.1	26	1	-	A	0.9	60	-	-	-
62	Ilavarasan	42/M	867415	+	+	-	-	-	+	-	-	-	Good	2	0.8	28	0.4	-	B	0.7	90	-	-	-
63	Xavier	46/M	882344	-	-	-	-	-	+	-	-	-	Excel	2.6	0.5	30	0.7	-	A	0.6	110	-	-	-
64	Rajendran	27/M	826773	-	-	-	-	-	+	-	-	-	Excel	3.2	0.8	31	0.6	-	A	0.8	90	-	-	-
65	Seetharaman	36/M	827793	-	+	-	-	-	+	-	-	-	Excel	3.1	0.8	29	0.8	-	A	0.9	100	-	-	-
66	Mohanraj	42/M	912191	-	-	-	-	-	+	-	-	-	Excel	2.9	0.7	30	0.6	-	A	0.8	90	-	-	-
67	Parimala	45/F	953949	-	-	-	--	-	+	-	-	-	Excel	3	0.9	35	0.8	-	A	1.1	70	-	-	-

68	Selvakumar	41/M	841109	-	-	-	-	-	+	-	-	-	Excel	2.8	0.6	31	0.9	-	A	0.8	100	-	-	-
69	Kamala	53/F	840050	-	-	-	-	-	+	-	-	-	Good	3.1	0.9	33	1.1	-	B	1	130	-	-	E.coli
70	Suriyagandhi	35/F	830909	-	-	-	-	-	+	-	-	-	Excel	2.9	0.8	39	0.9	-	A	0.8	110	-	-	-
71	Seethalakshmi	40/F	867733	-	+	-	-	-	+	-	-	-	Excel	2.8	0.7	35	0.7	-	A	1	100	-	-	-
72	Arumugam	48/M	840890	-	-	-	-	-	+	-	-	-	Excel	3.2	0.9	40	0.8	-	A	0.8	90	-	-	-
73	Ponnuram	42/M	830574	-	-	-	-	-	+	-	-	-	Good	3.1	0.6	35	0.6	-	B	0.9	70	-	-	-
74	Chandrasekar	39/M	882491	-	-	-	-	-	+	-	-	-	Excel	3	0.7	33	0.8	-	A	1.1	80	-	-	-
75	Kamalakannan	46/M	827469	-	-	-	-	-	+	-	-	-	Excel	2.9	0.6	30	0.6	-	A	1	100	-	-	-